

Neurofeedback for Attention-Deficit/Hyperactivity Disorder

A Systematic Review and Meta-Analysis

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 Supplemental content

IMPORTANCE Neurofeedback has been proposed for the treatment of attention-deficit/hyperactivity disorder (ADHD) but the efficacy of this intervention remains unclear.

OBJECTIVE To conduct a meta-analysis of randomized clinical trials (RCTs) using probably blinded (ie, rated by individuals probably or certainly unaware of treatment allocation) or neuropsychological outcomes to test the efficacy of neurofeedback as a treatment for ADHD in terms of core symptom reduction and improved neuropsychological outcomes.

DATA SOURCES PubMed (MEDLINE), Ovid (PsycInfo, MEDLINE, Embase + Embase Classic), and Web of Science, as well as the reference lists of eligible records and relevant systematic reviews, were searched until July 25, 2023, with no language limits.

STUDY SELECTION Parallel-arm RCTs investigating neurofeedback in participants of any age with a clinical ADHD or hyperkinetic syndrome diagnosis were included.

DATA EXTRACTION AND SYNTHESIS Standardized mean differences (SMDs) with Hedges *g* correction were pooled in random effects meta-analyses for all eligible outcomes.

MAIN OUTCOMES AND MEASURES The primary outcome was ADHD total symptom severity assessed at the first postintervention time point, focusing on reports by individuals judged probably or certainly unaware of treatment allocation (probably blinded). Secondary outcomes were inattention and/or hyperactivity-impulsivity symptoms and neuropsychological outcomes postintervention and at a longer-term follow-up (ie, after the last follow-up time point). RCTs were assessed with the Cochrane risk of bias tool version 2.0.

RESULTS A total of 38 RCTs (2472 participants aged 5 to 40 years) were included. Probably blinded reports of ADHD total symptoms showed no significant improvement with neurofeedback ($k = 20$; $n = 1214$; SMD, 0.04; 95% CI, -0.10 to 0.18). A small significant improvement was seen when analyses were restricted to RCTs using established standard protocols ($k = 9$; $n = 681$; SMD, 0.21; 95% CI, 0.02 to 0.40). Results remained similar with adults excluded or when analyses were restricted to RCTs where cortical learning or self-regulation was established. Of the 5 neuropsychological outcomes analyzed, a significant but small improvement was observed only for processing speed ($k = 15$; $n = 909$; SMD, 0.35; 95% CI, 0.01 to 0.69). Heterogeneity was generally low to moderate.

CONCLUSIONS AND RELEVANCE Overall, neurofeedback did not appear to meaningfully benefit individuals with ADHD, clinically or neuropsychologically, at the group level. Future studies seeking to identify individuals with ADHD who may benefit from neurofeedback could focus on using standard neurofeedback protocols, measuring processing speed, and leveraging advances in precision medicine, including neuroimaging technology.

JAMA Psychiatry. 2025;82(2):118-129. doi:10.1001/jamapsychiatry.2024.3702
Published online December 11, 2024.

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Attention-deficit/hyperactivity disorder (ADHD) is a neurodevelopmental condition characterized by developmentally inappropriate, persistent, pervasive, and impairing inattention and/or hyperactivity-impulsivity.¹ ADHD medications, particularly psychostimulants, reduce symptoms and impairment at least in the short term^{2,3} and are recommended as part of multimodal treatment approaches, including psychosocial therapies or psychoeducation programs.^{3,4}

Neurofeedback has been proposed as a nonpharmacological treatment for ADHD. It deploys a brain-computer interface to train self-regulation of brain activity using real-time audiovisual feedback of brain activity.⁵ Electroencephalogram (EEG)-based neurofeedback is the most common, while newer techniques training hemodynamic brain signals for deeper and more focal regulation are being explored (eg, real-time functional magnetic resonance imaging [rt-fMRI] neurofeedback and functional near infrared spectroscopy [fNIRS] neurofeedback).⁶⁻¹¹ Two EEG neurofeedback protocols have typically been used. Frequency band training aims to decrease the power of θ activity (4-7 Hz) or increase the β activity (13-30 Hz) power to promote attention. Event-related slow cortical potential regulation aims to increase or decrease negative potential shifts linked to cortical activation and cognitive preparation.¹²⁻¹⁵ Building on evidence from learning theories, such as operant conditioning (ie, implicit learning through reinforcement) or skill acquisition (ie, explicit, goal-directed learning), neurofeedback protocols are premised on the notion that by learning to self-regulate or normalize ADHD-related brain activity, one can alleviate behavioral symptoms and related impairments.^{13,16}

However, the clinical efficacy of neurofeedback as a treatment for ADHD remains disputed despite nearly 5 decades of research. Randomized clinical trials (RCTs) have often been of poor quality due to inadequate blinding after treatment allocation (eg, use of unblinded parent reports or lack of clarity of blinding status), use of nonstandardized suboptimal treatment protocols, or failure to assess self-regulation or learning.¹⁶⁻¹⁹

In 2013, the European ADHD Guidelines Group (EAGG) published a meta-analysis,²⁰ followed by an update²¹ published in 2016, of RCTs of EEG neurofeedback in children and adolescents with ADHD. A central feature of these meta-analyses was the exploration of the impact of blinding by contrasting 2 types of ADHD symptom outcomes: those reported by individuals most proximal to the intervention setting and so judged least likely to have been blinded (eg, parent reports on home implemented neurofeedback) and those reported by individuals confirmed as certainly blinded or who were probably unaware of the treatment allocation, either due to their remoteness from the treatment setting (eg, teacher reports for home-based treatment) or because they were independent observers (eg, clinicians not involved in treatment). The EAGG 2013 analysis, only powered to analyze ADHD total symptoms, showed a significant neurofeedback-related improvement based on most proximal reports (SMD, 0.59; 95% CI, 0.31 to 0.87) but this effect was halved and became nonsignificant with probably blinded reports (SMD, 0.29; 95% CI, -0.02 to 0.61), suggesting outcome assessor bias. The EAGG 2016

Key Points

Question Is neurofeedback an efficacious stand-alone treatment for attention-deficit/hyperactivity disorder (ADHD)?

Findings This systematic review and meta-analysis included 38 randomized clinical trials (RCTs) and a total of 2472 participants. The analyses with probably blinded reports or neuropsychological outcomes provided no evidence of meaningful benefits of neurofeedback as a treatment for ADHD; however, small but statistically significant positive effects were observed when the analyses were limited to RCTs using well-established standard neurofeedback protocols and specifically targeted processing speed as an outcome measure.

Meaning The findings suggest that RCTs using probably blinded outcomes do not support the use of neurofeedback as a stand-alone ADHD treatment.

analysis confirmed and extended these findings, showing significant but small neurofeedback-related reductions across core ADHD symptoms for most proximal reports (SMDs, 0.26 to 0.36) but not for probably blinded reports (SMDs, 0.06 to 0.17). Further, for RCTs using established standard neurofeedback protocols (as defined by Arns et al 2014¹⁵ and 2020¹⁶), most proximal effects sizes increased (SMDs, 0.45 to 0.55) but probably blinded outcomes could not be analyzed due to insufficient data. Neurofeedback did not improve neuropsychological functioning. In short, EAGG meta-analytic evidence does not support neurofeedback as a stand-alone treatment for ADHD. Broadly similar results have been found in other meta-analyses of blinded RCTs of neurofeedback for ADHD.^{14,22-29}

A considerable number of neurofeedback RCTs have been published since the last search conducted by the EAGG in 2015. Many RCTs now include larger samples, longer-term follow-up periods, well-controlled designs, and more have probably blinded outcomes. Some have tested the efficacy of rt-fMRI neurofeedback or fNIRS neurofeedback. In light of this, we present an updated meta-analysis of neurofeedback RCTs for ADHD to provide a more accurate and comprehensive estimate of the value of neurofeedback as a stand-alone treatment for ADHD in terms of core symptom reduction and improved neuropsychological performance. We also conducted sensitivity analyses with probably blinded outcomes relating to whether effects were stronger when standard EEG neurofeedback protocols were followed and when there was evidence that learning or self-regulation had occurred. We also ran exploratory analyses for individuals most proximal to the intervention to see if novel neurofeedback approaches targeting hemodynamic signals of brain activity were efficacious and to compare neurofeedback to medication and other nonpharmacological interventions in head-to-head trials.

Methods

This preregistered systematic review and meta-analysis (CRD42022290005) was reported according to Preferred Reporting Items for Systematic Reviews and Meta-analyses

(PRISMA)³⁰ and PRISMA-S³¹ reporting guidelines (eAppendix in Supplement 1).

Search Strategy

PubMed (MEDLINE), Ovid (PsycInfo, Medline, Embase + Embase Classic), and Web of Science were searched until July 25, 2023 (eMethods 3 in Supplement 1). All articles before this search date were de novo screened for eligibility. No language limits were imposed.

Eligibility and Selection

We included only peer-reviewed, published parallel-arm RCTs where participants had a clinical ADHD or hyperkinetic syndrome diagnosis as defined by *DSM-III/ICD-9* onward (any subtype or presentation) or with above cutoff scores on validated ADHD rating scales (eMethods 1 and 3 in Supplement 1). Disagreements were resolved by senior authors (D.B., S.C., and E.S.B.). Authors of RCTs were contacted for unpublished data or information as needed. RCTs were assessed with the Cochrane risk of bias tool version 2.0.

Data Extraction

We extracted outcome means and standard deviations at all available time points. Where multiple ADHD outcomes were reported, only one was selected according to a prespecified hierarchy (eMethods 4 in Supplement 1). Outcomes for individuals most proximal to the intervention included parent ratings (including when reported by a clinician) if the intervention was home based, teacher ratings if school based, investigators or clinicians if lab or clinic based, or self-ratings by adults regardless of intervention setting. Probably blinded estimates included reports given by individuals judged as probably unaware of the treatment allocation because of their remoteness from the trial setting, independent observers, or a certainly blinded self- or parent-rating in an RCT with a sham control arm. If multiple probably blinded reports were available, the outcome judged best blinded was selected (ie, independent assessors; eg, through direct observation) or the most remote assessor from the intervention setting (eg, teacher over parent if home based). While outcomes for individuals most proximal to the intervention were, by definition, available from all RCTs with ADHD-related outcomes, fewer probably blinded outcomes were identified. The most proximal and probably blinded outcomes were the same (ie, from 1 blinded assessor) for only Alegria et al^{11,32-34} (2017), Baumeister et al³⁵ (2018), Bink et al³⁶⁻³⁸ (2014, 2015, 2016), Lam et al⁹ (2022), and Zilverstand et al¹⁰ (2017).

Statistical Analysis

Our primary outcome was ADHD core symptom severity (total combined; ie, inattention and hyperactivity-impulsivity) measured at the first time point after the final neurofeedback session. Secondary outcomes were inattention and hyperactivity-impulsivity symptoms (separately) or neuropsychological outcomes at the first time point after the final neurofeedback session or at a longer-term follow-up assessment (≥ 3 months and the last follow-up time point). Neuropsychological outcomes were grouped as per our previous meta-analysis.³⁹

Effect size estimates (standardized mean differences [SMDs]) were calculated as mean baseline to postassessment (or follow-up) change in the intervention group minus the mean baseline to postassessment (or follow-up) change in the control group divided by the pooled baseline standard deviation, with Hedges *g* small sample bias adjustment.^{40,41} We conducted random effects models meta-analyses for all outcomes at all available time points (ie, baseline vs postassessment or follow-up). SMDs were combined using the inverse variance method.^{40,42} Between-SMD heterogeneity was tested using the χ^2 (Q) test, while the magnitude of true vs random heterogeneity was estimated using the I^2 statistic.⁴⁰ As in previous EAGG meta-analyses,^{20,39,43,44} at least 5 relevant RCTs were required per outcome domain to reduce between-SMD heterogeneity.⁴⁵

Sensitivity analyses were conducted to examine neurofeedback efficacy for slow cortical potential regulation and frequency band training EEG neurofeedback separately, when an established standard protocol was used as defined by Arns et al 2014¹⁵ and 2020¹⁶ (eMethods 5 in Supplement 1), when neurofeedback learning was demonstrated (defined as statistically significant change in EEG or hemodynamic patterns in the expected direction), when probably blinded assessments were made in the intervention setting (eg, direct observation by an independent observer), when participants were children or adolescents only (aged <18 years), and when the comparator arms were sham or semiactive controls (eg, EMG training), placebo tablet, treatment as usual, and waitlist control. Meta-regressions were conducted if at least 10 RCTs per predictor (as suggested by Borenstein and Higgins⁴⁶) were available, with the following predictors: publication year, mean age of sample, percentage of participants medicated at baseline during the treatment period, and overall risk of bias (low, some concerns, or high).⁴⁷ Additional unregistered exploratory analyses were conducted for RCTs of novel neurofeedback targeting hemodynamic signals and head-to-head RCTs comparing neurofeedback with medication or other nonpharmacological treatments.

Publication bias was assessed using the Egger regression test of small study effects, but only for significant results from the main analysis without significant heterogeneity. Data analysis and data visualization were conducted in RevMan version 5 (Cochrane)⁴² and the metafor package in RStudio 2024.09.0 build 375(R Foundation).⁴⁸

Results

Study Characteristics

From an initial 1457 records, we retained 38 RCTs reported in 53 publications (2472 participants aged 5 to 40 years) (Table 1) (see eFigure 1 in Supplement 1 for the PRISMA flowchart; for included and excluded reports and outcomes, see eMethods 6 and eTables 1-3 in Supplement 1). Thirty-three RCTs applied EEG neurofeedback (frequency band training, 24; slow cortical potential regulation, 8; frequency band training plus slow cortical potential regulation). Five applied fMRI or fNIRS. Three studied adults. Nine reported eligible longer-term outcomes

Table 1. Summary of Results Showing Pooled Standardized Mean Differences (SMDs) Between Treatment and Control Arms for Probably Blinded Measures of Attention-Deficit/Hyperactivity Disorder Symptoms at the First Assessment After the Final Neurofeedback Session

Probably blinded outcome	RCTs included	RCTs, No.	Participants, No.	Effect size estimate, SMD (95% CI) ^a	P value	Heterogeneity		
						Q	I ² , %	P value
ADHD total	All	20	1214	0.04 (−0.10 to 0.18)	.56	27.37	31	.10
	EEG only	15	1038	0.09 (−0.07 to 0.25)	.29	21.98	36	.08
	FBT	9	609	0.02 (−0.22 to 0.27)	.85	15.82	49	.04
	SCP	4	NA	NA	NA	NA	NA	NA
	Standard protocol	9	681	0.21 (0.02 to 0.40)	.03	12.96	38	.11
	Hemodynamic only	5	176	−0.15 (−0.42 to 0.11)	.26	3.03	0	.55
	Active, nonpharmacological	2	NA	NA	NA	NA	NA	NA
	Sham, semiactive	14	783	0.05 (−0.09 to 0.20)	.45	14.44	10	.34
	TAU	2	NA	NA	NA	NA	NA	NA
	WLC	2	NA	NA	NA	NA	NA	NA
	With learning	5	238	0.23 (−0.03 to 0.49)	.08	2.20	0	.70
	Children only	18	1126	0.05 (−0.10 to 0.20)	.50	26.11	35	.07
	Same intervention and probably blinded setting	2	NA	NA	NA	NA	NA	NA
Inattention	All	19	1176	0.04 (−0.15 to 0.22)	.69	38.71	54	.003
	EEG only	14	1000	0.12 (−0.07 to 0.30)	.23	24.84	48	.02
	FBT	9	609	0.11 (−0.16 to 0.38)	.41	18.38	57	.02
	SCP	3	NA	NA	NA	NA	NA	NA
	Standard protocol	8	643	0.22 (−0.03 to 0.47)	.08	15.40	55	.03
	Hemodynamic only	5	176	−0.29 (−0.69 to 0.11)	.15	5.98	33	.20
	Active, nonpharmacological	1	NA	NA	NA	NA	NA	NA
	Sham, semiactive	14	783	−0.01 (−0.21 to 0.20)	.96	23.46	45	.04
	TAU	2	NA	NA	NA	NA	NA	NA
	WLC	2	NA	NA	NA	NA	NA	NA
	With learning	5	238	0.22 (−0.13 to 0.58)	.21	5.50	27	.24
	Children only	17	1088	0.04 (−0.16 to 0.23)	.71	35.26	55	.004
	Same intervention and probably blinded setting	2	NA	NA	NA	NA	NA	NA
Hyperactivity/impulsivity	All	19	1176	0.03 (−0.09 to 0.16)	.59	22.15	19	.23
	EEG only	14	1000	0.06 (−0.10 to 0.22)	.45	21.32	39	.07
	FBT	9	609	0.03 (−0.19 to 0.24)	.80	14.10	43	.08
	SCP	3	NA	NA	NA	NA	NA	NA
	Standard protocol	8	643	0.13 (−0.04 to 0.30)	.14	10.10	31	.18
	Hemodynamic only	5	176	−0.06 (−0.36 to 0.24)	.70	0.43	0	.98
	Active, nonpharmacological	1	NA	NA	NA	NA	NA	NA
	Sham, semiactive	14	783	0.08 (−0.04 to 0.20)	.19	7.39	0	.88
	TAU	2	NA	NA	NA	NA	NA	NA
	WLC	2	NA	NA	NA	NA	NA	NA
	With learning	5	238	0.17 (−0.05 to 0.39)	.12	4.14	3	.39
	Children only	17	1088	0.05 (−0.09 to 0.19)	.51	21.84	27	.15
	Same intervention and probably blinded setting	2	NA	NA	NA	NA	NA	NA

Abbreviations: EEG, electroencephalogram; FBT, frequency band training; NA, not applicable; SCP, slow cortical potentials; TAU, treatment as usual; WLC, waitlist control.

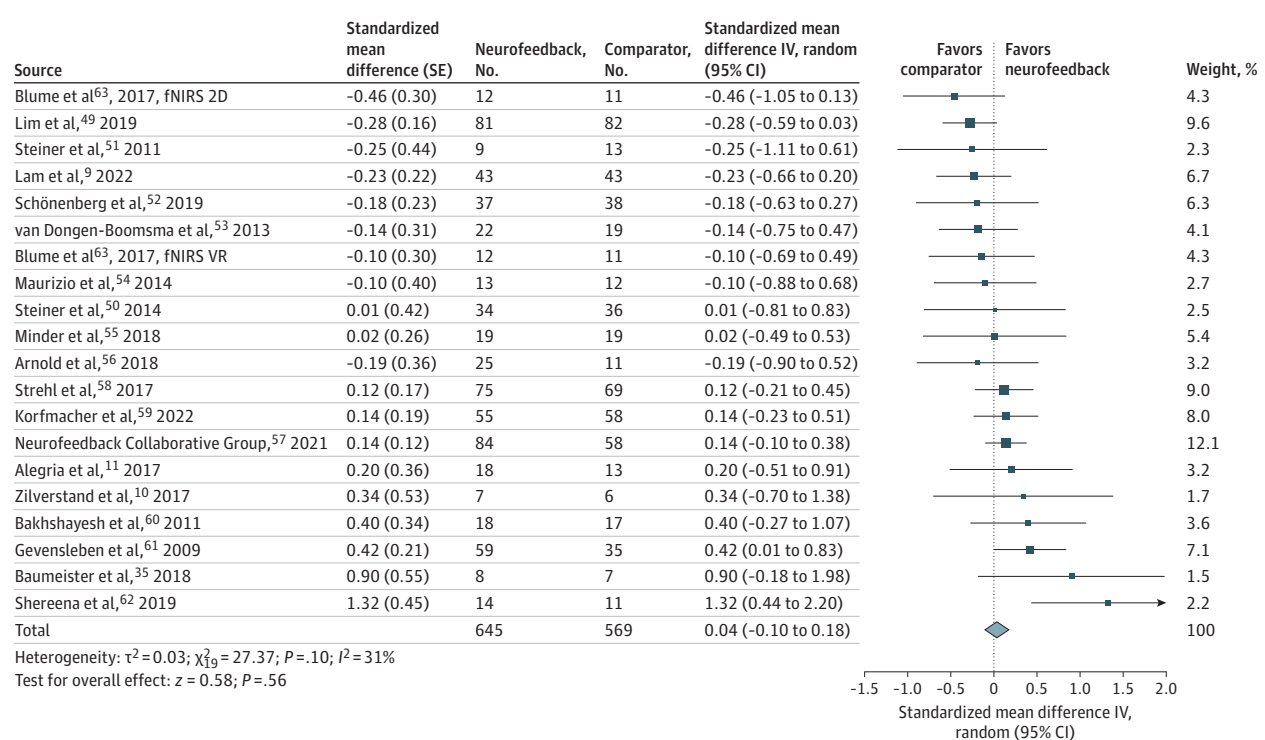
^a With Hedges *g* adjustment.

(ie, >3 months after neurofeedback was completed; range 6-12 months). Forty-two RCTs were rated as overall high risk of bias, mainly due to inadequate blinding (eFigure 2 in [Supplement 1](#)).

Meta-Analysis Results

We report below the results from our analysis of trials with probably blinded outcomes^{9-11,35,49-62} for a more unbiased and robust estimate of neurofeedback effects. For fNIRS or fMRI

Figure 1. Meta-Analysis of Effects of Probably Blinded Outcome Measures of Attention-Deficit/Hyperactivity Disorder—Total Symptoms



IV indicates inverse variance; random, random-effects meta-analysis.

neurofeedback or head-to-head trials, we used most proximal reports due to insufficient, probably blinded data (<5 trials). Other analyses of most proximal outcomes are provided in eTables 4, 8, and 10 in [Supplement 1](#).

Primary Outcome Measure

There was no significant neurofeedback-related improvement on total probably blinded ADHD symptoms ($k=20$; $n=1214$; SMD, 0.04; 95% CI, -0.10 to 0.18) (Table 1 and [Figure 1](#)⁶³; see [eFigure 3](#) and [eTable 4](#) in [Supplement 1](#) for most proximal outcomes). This finding was replicated across nearly all sensitivity analyses ([Figure 1](#) and Table 1; [eTable 5](#) in [Supplement 1](#)). A statistically significant but small effect of neurofeedback was found in RCTs using standard neurofeedback protocols ($k=9$; $n=681$; SMD, 0.21; 95% CI, 0.02 to 0.40). Heterogeneity was generally low and nonsignificant (Table 1).

Secondary Outcome Measures

ADHD Subdimensions

There were no significant reductions in probably blinded inattention or hyperactivity-impulsivity symptoms (replicated in all sensitivity analyses). Heterogeneity was generally moderate and significant for inattention and low and nonsignificant for hyperactivity-impulsivity ([Figure 2](#) and Table 1; [eTables 4-5](#) in [Supplement 1](#)).

Neuropsychological Outcomes

Neurofeedback significantly improved speed of processing ($k=15$; $n=909$; SMD, 0.35; 95% CI, 0.01-0.69), but not atten-

tion, inhibition, verbal working memory, or visual working memory. Heterogeneity was generally high and significant (Table 2; [eTable 6](#) in [Supplement 1](#)).

Longer-Term Follow-Up Outcomes

There were no significant longer-term effects of neurofeedback in analyses with all RCTs included (mean [range], 6 [6-12] months) ([eTables 7-9](#) in [Supplement 1](#)), with the exception of a significant improvement for processing speed ($k=8$; $n=517$; SMD, 0.32; 95% CI, 0.04-0.61), although this was associated with significant heterogeneity.

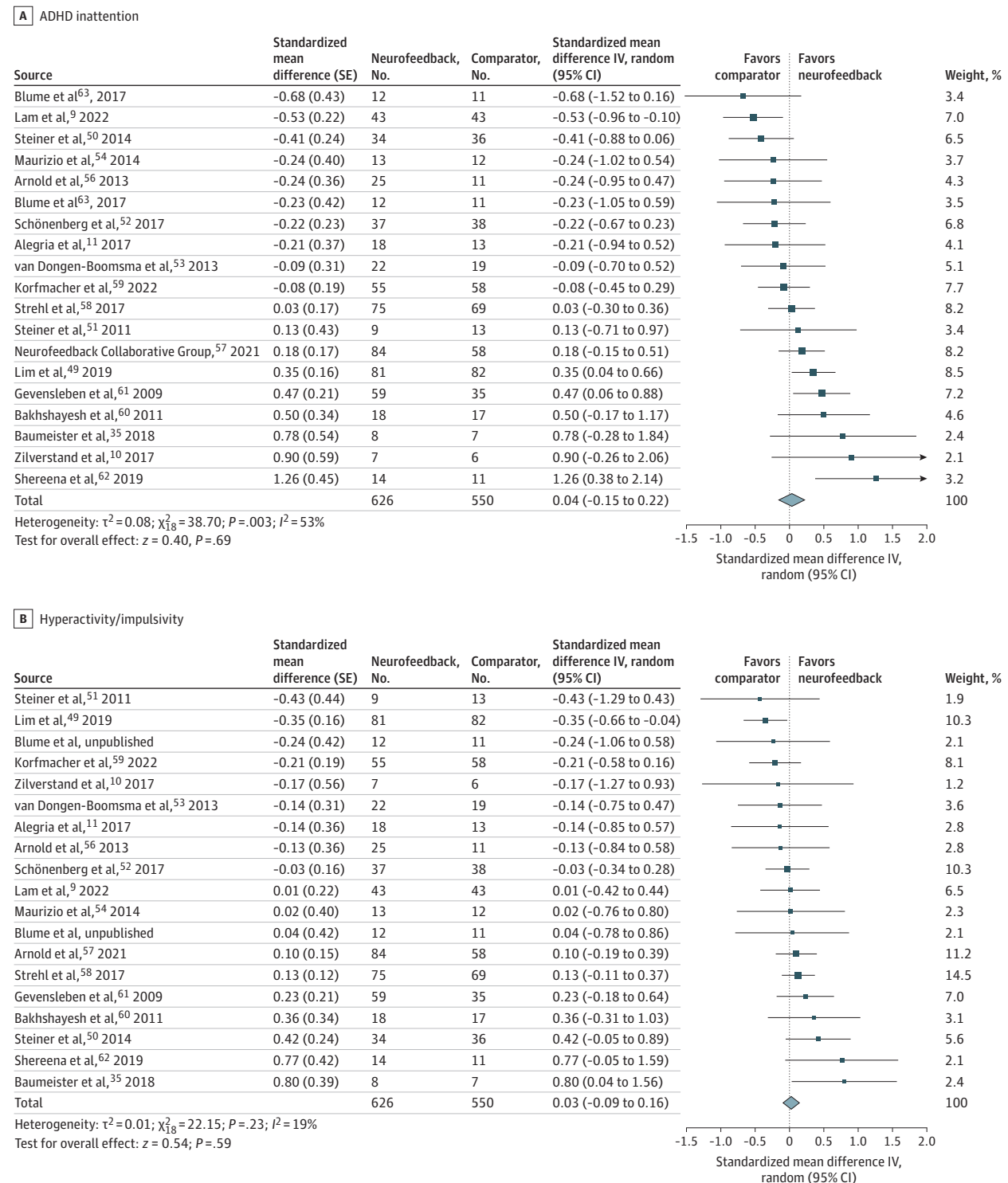
Meta-Regression

Neurofeedback effect sizes were generally not significantly predicted by mean age, percentage of medicated participants, publication year, risk of bias, or activeness of comparator arms ([eTable 10](#) in [Supplement 1](#)).

Exploratory Analysis

Neurofeedback vs Other Treatments

Most proximal reports of ADHD symptoms showed methylphenidate had a significantly greater effect than neurofeedback on total ADHD ($k=7$; $n=417$; SMD, -0.68; 95% CI, -0.92 to -0.44), inattention ($k=7$; $n=418$; SMD, -0.74; 95% CI, -0.9 to -0.52), and hyperactivity-impulsivity symptoms ($k=7$; $n=417$; SMD, -0.49; 95% CI, -0.73 to -0.26). There was no significant difference between neurofeedback and other non-pharmacological interventions (ie, cognitive training and physical exercise). All analyses showed low and nonsignificant

Figure 2. Meta-Analysis of Effects of Probably Blinded Outcome Measures of Attention-Deficit/Hyperactivity Disorder—Inattention and Hyperactivity/Impulsivity Symptoms

heterogeneity (eTable 11 in Supplement 1). There were insufficient head-to-head trials to analyze probably blinded outcomes.

fMRI and fNIRS

No benefits were observed with fMRI and fNIRS interventions for most proximal reports of ADHD symptoms (Tables 1

Table 2. Summary of Results Showing Pooled Standardized Mean Differences (SMDs Between Treatment and Control Arms for Neuropsychological Measures at the First Assessment After the Final Neurofeedback Session

Outcome	RCTs included	RCTs, No.	Participants, No.	Effect size estimate, SMD (95% CI) ^a	P value	Heterogeneity		
						Q	I ² , %	P value
Attention	All	25	1140	0.04 (−0.19 to 0.26)	.75	76	68	<.001
	EEG only	19	922	0.11 (−0.15 to 0.37)	.40	64	72	<.001
	FBT	11	444	0.38 (−0.05 to 0.81)	.08	45	78	<.001
	SCP	5	260	−0.27 (−0.63 to 0.08)	.13	7	42	.14
	Standard protocol	15	687	0.18 (−0.16 to 0.52)	.29	61	77	<.001
	Hemodynamic only	6	218	−0.21 (−0.63 to 0.20)	.31	10	51	.07
	Active, nonpharmacological	3	NA	NA	NA	NA	NA	NA
	Sham, semiactive	16	687	0.06 (−0.30 to 0.41)	.76	72	79	<.001
	TAU	4	NA	NA	NA	NA	NA	NA
	WLC	2	NA	NA	NA	NA	NA	NA
	With learning	7	247	0.35 (−0.55 to 1.25)	.45	58	90	<.001
	Children only	22	1040	0.03 (−0.22 to 0.28)	.81	75	72	<.001
Inhibition	All	21	990	0.08 (−0.10 to 0.26)	.39	48	59	<.001
	EEG only	15	778	0.04 (−0.16 to 0.24)	.72	26	45	.03
	FBT	8	335	0.22 (−0.12 to 0.56)	.20	16	56	.03
	SCP	5	260	−0.07 (−0.35 to 0.20)	.61	5	14	.32
	Standard protocol	13	610	0.06 (−0.18 to 0.31)	.61	25	52	.01
	Hemodynamic only	6	212	0.20 (−0.23 to 0.64)	.36	21	76	.001
	Active, nonpharmacological	3	NA	NA	NA	NA	NA	NA
	Sham, semiactive	12	538	0.05 (−0.21 to 0.31)	.69	32	66	.001
	TAU	4	NA	NA	NA	NA	NA	NA
	WLC	2	NA	NA	NA	NA	NA	NA
	With learning	5	171	0.41 (−0.14 to 0.96)	.14	22	81	<.001
	Children only	18	890	0.12 (−0.11 to 0.35)	.29	43	61	<.001
Verbal WM	All	9	455	−0.04 (−0.19 to 0.11)	.58	4	0	.85
	EEG only	7	401	−0.05 (−0.21 to 0.11)	.53	4	0	.69
	FBT	4	NA	NA	NA	NA	NA	NA
	SCP	2	NA	NA	NA	NA	NA	NA
	Standard protocol	4	NA	NA	NA	NA	NA	NA
	Hemodynamic only	2	NA	NA	NA	NA	NA	NA
	Active, nonpharmacological	0	NA	NA	NA	NA	NA	NA
	Sham, semiactive	4	NA	NA	NA	NA	NA	NA
	TAU	3	NA	NA	NA	NA	NA	NA
	WLC	2	NA	NA	NA	NA	NA	NA
	With learning	1	NA	NA	NA	NA	NA	NA
	Children only	6	357	−0.14 (−0.39 to 0.12)	.30	8	35	.17

(continued)

and 2; eTables 4 and 7-9 in [Supplement 1](#)). There were insufficient trials to analyze probably blinded outcomes.

Discussion

In this systematic review and meta-analysis of RCTs focusing on at least probably blinded neuropsychological outcomes, we found no evidence to support the use of current forms of neurofeedback as stand-alone treatment for ADHD. This gener-

ally negative picture had 2 possible exceptions. The first is that established standard protocols led to a nominally significant benefit on probably blinded measures of total ADHD symptoms. However, 4 observations temper the importance of this finding. First, it could be due to the standard nature of the protocols or the mixture of different neurofeedback approaches that were analyzed (frequency band training vs slow cortical potential regulation). Second, the small improvement (SMD, 0.21) likely falls short of clinical value and is smaller than what we found in 2016 with only 3 trials available for analysis (SMD,

Table 2. Summary of Results Showing Pooled Standardized Mean Differences (SMDs Between Treatment and Control Arms for Neuropsychological Measures at the First Assessment After the Final Neurofeedback Session (continued))

Outcome	RCTs included	RCTs, No.	Participants, No.	Effect size estimate, SMD (95% CI) ^a	P value	Heterogeneity		
						Q	I ² , %	P value
Visual WM	All	7	449	−0.10 (−0.24 to 0.04)	.17	3	0	.79
	EEG only	7	349	−0.10 (−0.25 to 0.06)	.21	3	0	.64
	FBT	3	NA	NA	NA	NA	NA	NA
	SCP	1	NA	NA	NA	NA	NA	NA
	Standard protocol	3	NA	NA	NA	NA	NA	NA
	Hemodynamic only	2	NA	NA	NA	NA	NA	NA
	Active, nonpharmacological	0	NA	NA	NA	NA	NA	NA
	Sham, semiactive	4	NA	NA	NA	NA	NA	NA
	TAU	3	NA	NA	NA	NA	NA	NA
	WLC	0	NA	NA	NA	NA	NA	NA
	With learning	1	NA	NA	NA	NA	NA	NA
	Children only	6	436	−0.11 (−0.25 to 0.04)	.14	3	0	.75
Processing speed	All	15	909	0.35 (0.01 to 0.69)	.04	85	84	<.001
	EEG only	13	909	0.35 (0.01 to 0.69)	.04	85	84	<.001
	FBT	8	365	0.67 (−0.06 to 1.39)	.07	72	90	<.001
	SCP	3	909	0.35 (0.01 to 0.69)	.04	85	84	<.001
	Standard protocol	11	641	0.48 (0.00 to 0.95)	.05	80	87	<.001
	Hemodynamic only	2	NA	NA	NA	NA	NA	NA
	Active, nonpharmacological	2	NA	NA	NA	NA	NA	NA
	Sham, semiactive	11	632	0.57 (0.11 to 1.02)	.01	73	86	<.001
	TAU	2	NA	NA	NA	NA	NA	NA
	WLC	2	NA	NA	NA	NA	NA	NA
	With learning	4	NA	NA	NA	NA	NA	NA
	Children only	12	747	0.34 (−0.07 to 0.75)	.11	79	86	<.001
BRIEF-GEC score	All	7	385	0.13 (−0.04 to 0.30)	.13	3	0	.81
	EEG only	7	385	0.13 (−0.04 to 0.30)	.13	3	0	.81
	FBT	3	NA	NA	NA	NA	NA	NA
	SCP	2	NA	NA	NA	NA	NA	NA
	Standard protocol	2	NA	NA	NA	NA	NA	NA
	Hemodynamic only	0	NA	NA	NA	NA	NA	NA
	Active, nonpharmacological	1	NA	NA	NA	NA	NA	NA
	Sham, semiactive	2	NA	NA	NA	NA	NA	NA
	TAU	3	324	0.14 (−0.04 to 0.33)	.13	3	0	.60
	WLC	1	324	0.14 (−0.04 to 0.33)	.13	3	0	.60
	With learning	0	NA	NA	NA	NA	NA	NA
	Children only	7	385	0.13 (−0.04 to 0.30)	.13	3	0	.81

Abbreviations: BRIEF-GEC, Behavior Rating Inventory of Executive Function-Global Executive Composite; EEG, electroencephalogram; FBT, frequency band training; NA, not applicable; SCP, slow cortical potentials;

TAU, treatment as usual; WLC, waitlist control; WM, working memory.

^a With Hedges *g* adjustment.

0.36).²⁰ Third, the improvement was not observed in probably blinded measures of inattention or hyperactivity-impulsivity symptoms, separately. Fourth, the longer-term persistence of this improvement is unclear. Insufficient trials prevented an analysis of longer-term probably blinded outcomes while outcomes for individuals most proximal to the intervention showed no significant improvement. The second exception is that neurofeedback improved processing speed, a core neu-

ropsychological component that may underpin higher-order cognitive impairment in ADHD^{64,65}—an effect that was also found in longer-term follow-up periods. The clinical value of these improvements is questionable, as they were barely significant with significant heterogeneity. Furthermore, although effect size estimates were small to moderate, they were derived from analyses with multiple dependent effect size estimates (6 of 15 in the postassessment analysis and 6 of 8 in

the follow-up analysis) across 3 RCTs with overlapping comparator samples, potentially inflating the observed effects.

Several of our findings warrant discussion. First, methylphenidate was superior to neurofeedback in alleviating core ADHD symptoms based on outcomes for individuals most proximal to the intervention (but not probably blinded outcomes due to insufficient data). The estimated effect sizes ($k = 7$; SMD range, -0.49 to -0.74 ; 95% CI range, -0.73 to -0.23) were similar to those from meta-analyses³ of RCTs comparing methylphenidate with placebo in ADHD (SMD range, -0.49 to -0.82 ; 95% CI range, -1.16 to -0.62), indicating limited clinical benefit of neurofeedback. We also found no significant difference between neurofeedback and other nonpharmacological treatments (ie, cognitive training and exercise), suggesting a general ineffectiveness of nonpharmacological interventions for ADHD core symptoms when considering outcomes rated by blinded individuals. This aligns with our recent meta-analytical evidence showing negligible efficacy of cognitive training for ADHD.³⁹ Second, although no benefits were found with hemodynamic neurofeedback techniques (eg, fMRI and fNIRS), this is based on only 5 trials; further research is warranted given the potential to target deeper ADHD-relevant brain regions (eg, opercular right inferior frontal cortex or basal ganglia) with greater spatial (but not temporal) resolution than EEG-based approaches.⁹ Third, no significant clinical improvements were found at longer-term follow-up points (ie, 6 to 12 months after neurofeedback training). Fourth, effect sizes for probably blinded and most proximal outcomes were smaller in the current than in the 2016 meta-analysis (SMD ranges: probably blinded, 0.03 to 0.04 vs 0.06 to 0.15 and most proximal, 0.26 to 0.36 vs 0.19 to 0.26), but meta-regressions showed no significant relationship between publication year and effect size, suggesting factors other than improved trial design caused this reduction over time.

From a methodological point of view, two findings are noteworthy. First, neurofeedback effects did not vary as a function of how active the comparator arm was, as effects were not observed even in trials with less active control conditions. However, the limited number of trials with the least active and nonpharmacological alternative control conditions weakens this conclusion. Second, there was no relationship between whether an RCT established the existence of neurofeedback learning and clinical improvement in ADHD symptoms, suggesting that previous evidence of clinical improvement could in this situation potentially result from nonspecific, incidental impacts of neurofeedback protocols (eg, wraparound treatment, skill development, and improving tolerance of effortful tasks) rather than self-regulation of brain activity or that the relationship holds only for learning subgroups.⁶⁶ However, this interpretation is based only on 5 RCTs, so the mechanisms driving self-regulation and the individual clinical effects need further investigation.

Given these negative results, what is the future of neurofeedback as a treatment for ADHD? First, the limited but significant improvements with standard neurofeedback protocols and in processing speed needs further validation through methodologically sound trials, as this could help refine future approaches targeting higher-order functions.¹³ Second, although RCTs provide aggregate-level data, an individu-

alized neurofeedback approach could be developed by tailoring neurofeedback parameters to individual characteristics. However, reliable baseline indicators (eg, demographic, neuropsychological, and brain function differences) of potential responders to specific training regimes (eg, slow cortical potential regulation training depending on baseline slow cortical potentials) are currently lacking.¹³ We only had sufficient RCTs to show that baseline medication status did not predict neurofeedback effects. Third, combining neurofeedback with noninvasive brain stimulation techniques (eg, repetitive transcranial magnetic stimulation, transcranial direct current stimulation, and external trigeminal nerve stimulation) could be potentially fruitful, but evidence supporting their efficacy when applied without neurofeedback is limited.⁶⁷⁻⁶⁹ Additionally, incorporating virtual reality or transfer trials (where no immediate feedback is given) could provide a more ecologically valid means to self-regulate symptoms in everyday life, but this approach remains underexplored.⁷⁰

Limitations

As with any meta-analysis, the scope of this one is limited by the trials available for inclusion. First, 1 in 2 trials with probably blinded reports measured self-regulation success, so we cannot rule out that the improvements we found might result from nonspecific psychological or behavioral effects rather than deliberate training of target brain activity. Testing whether self-regulation occurred and its relation to outcomes should be standard in future RCTs. Second, very few trials measured everyday functioning, quality of life, and academic outcomes, so the impact of neurofeedback beyond core symptoms and neuropsychological functioning remain uncertain. Third, we could not explore if probably blinded effects were setting specific, as most trials applied neurofeedback in a setting different from the assessment. Fourth, future trials should systematically measure longer-term (ie, beyond 3 months) effects of neurofeedback with probably blinded reports, especially given neurofeedback may induce long-term plasticity changes and could have delayed effects.¹³ Fifth, most RCTs were judged to have a high risk of bias due to inadequate blinding. Future studies should be double blinded and assess blinding integrity. Sixth, as too few trials with probably blinded outcomes from adult samples were available for analysis, future trials should recruit from this age group, although we do not expect major departures from the limited effects we found in younger samples. Seventh, the limited benefit of neurofeedback we report raises important questions about the opportunity costs associated with its use and the potential iatrogenic effects from null effects (eg, disappointment and feelings of lack of control), none of which were considered in the included trials. As recommended when evaluating psychological therapies for ADHD,⁷¹ future trials should seriously consider these questions; otherwise, assessment of the clinical value of neurofeedback will remain constrained.

Conclusion

In conclusion, after decades of research, we found no group-level evidence supporting neurofeedback as a stand-alone

treatment for ADHD. Future studies should therefore weigh the cost and benefits of administering neurofeedback over other treatments. Advances in precision medicine, including brain

imaging techniques, might eventually identify specific individuals with ADHD for whom neurofeedback could be effective and safe.

ARTICLE INFORMATION

Accepted for Publication: September 12, 2024.

Published Online: December 11, 2024.
doi:10.1001/jamapsychiatry.2024.3702

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Merkl, Balia, Goujon, Bousquet, Capodiferro, Derks, Purper-Ouakil, Carucci, Holtmann, Brandeis, Cortese. **Drafting of the manuscript:** Westwood, Nagy, Bousquet, Sonuga-Barke. **Critical review of the manuscript for important intellectual content:** Westwood, Aggensteiner, Kaiser, Nagy, Donno, Merkl, Balia, Goujon, Capodiferro, Derks, Purper-Ouakil, Carucci, Holtmann, Brandeis, Cortese. **Statistical analysis:** Westwood, Goujon, Derks. **Administrative, technical, or material support:** Westwood, Aggensteiner, Kaiser, Nagy, Merkl, Goujon, Bousquet, Capodiferro, Brandeis, Cortese. **Supervision:** Westwood, Aggensteiner, Nagy, Purper-Ouakil, Carucci, Holtmann, Brandeis, Cortese, Sonuga-Barke.

Conflict of Interest Disclosures: Dr Nagy reported personal fees from Medice and Egis Pharmaceuticals and conference participation support from Medice outside the submitted work. Dr Donno reported serving as a subinvestigator in clinical trials sponsored by Lundbeck and as an independent rater in clinical trials sponsored by Servier, Acadia, and Bioproject. Dr Balia reported travel fees from Medice and collaboration on projects from the EU Seventh Framework Program and on clinical trials sponsored by Lundbeck, Otsuka, Janssen-Cilag, Angelini, Bioproject, and Acadia. Dr Purper-Ouakil reported nonfinancial support from HAC Pharma and personal fees from Medice outside the submitted work. Dr Carucci reported personal fees from Medice and Ecupharma and collaboration on projects from the EU Seventh Framework Programme, Era-Net Neuron, and connect4children and on clinical trials sponsored by Lundbeck, Otsuka, Janssen-Cilag, Angelini, Acadia, and Bioproject. Dr Holtmann reported personal fees from Medice, Hogrefe Publishers, Takeda, and Infectopharm outside the submitted work. Dr Brandeis reported having served as an unpaid scientific advisor of an EU-funded neurofeedback study. Dr Cortese reported personal fees from Medice, Association for Child and Adolescent Mental Health, personal fees from British Association of Psychopharmacology, and Canadian ADHD Resource Alliance outside the submitted work. Dr Sonuga-Barke reported speaker fees from Takeda and Medice and in-kind support from ObTech outside the submitted work as well as personal fees from *Journal of Child Psychology and Psychiatry* (editor in chief), Aarhus University (visiting professor), and Hong Kong University (visiting professor) outside the submitted work and was involved in the development of parent training interventions. No other disclosures were reported.

Group Information: A list of EAGG members appears in **Supplement 2**.

Data Sharing Statement: See **Supplement 3**.

Additional Contributions: We would like to thank all the authors who kindly provided additional methodological details or data.

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